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CHAPTER 39

Ophthalmologic Diseases in Small Pet Mammals

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RABBITS

Ophthalmic examination of rabbits can be performed easily.[43, 44] The eyes are laterally located and have a round pupil. Evaluation of a menace response is difficult, but most rabbits will react to bright light by squinting. The dorsal rectus muscle can usually be seen as a large striated band of tissue under the conjunctiva. Some rabbits do not respond to topical application of mydriatic agents because of the natural presence of atropinase. In these rabbits, the addition of 10% phenylephrine may help to obtain mydriasis. Rabbits have a merangiotic fundus. The well-myelinated optic nerve is present above the visual axis and has a deep optic cup. Retinal blood vessels are present in a linear streak medial and lateral to the optic nerve. An extensive venous plexus is present in the orbit. Tear production in rabbits can be measured by using Schirmer tear test strips. Average tear production is 5 mm/min (standard deviation, ±2.4 mm/min)[1]; however, very low values can be measured in some normal rabbits. Normal intraocular pressure measured by applanation tonometry is between 10 and 20 mm Hg.

The nasolacrimal system of rabbits has a single nasolacrimal punctum. The punctum is located in the ventral eyelid 3 mm from the eyelid margin, near the medial canthus and ventral to the lacrimal caruncle (Fig. 39-1).[5,24] The lacrimal sac is immediately rostral to the punctum and caudal to the nasolacrimal duct aperture. The nasolacrimal duct extends from the orbit to the nasal fossa and runs within the part of the maxilla that forms the lateral wall of the maxillary sinus.[24] Approximately 5 to 6 mm within the maxilla, the duct curves sharply and decreases in diameter.[5] At the level of the palatine bone, the nasolacrimal duct leaves the bony nasolacrimal canal and makes a sharp turn at the nasolacrimal duct flexure, which is located just caudal to the caudal limit of the incisor tooth roots. The nasolacrimal duct narrows at this flexure in normal rabbits. The duct then follows the ventral margin of the nasoturbinates and exits on the ventromedial aspect of the alar fold just caudal to the mucocutaneous junction of the nares.

Conjunctivitis and Epiphora

Conjunctivitis in rabbits is common. In normal rabbits with no ocular or respiratory disease, the most frequently isolated organisms from the conjunctival cul de sac include *Bacillus subtilis, Staphylococcus aureus, Bordetella* species, and *Pasteurella* species. However, *Pasteurella multocida* is a cause of conjunctivitis, epiphora, nasolacrimal duct obstruction, and dacryocystitis in rabbits.[24] Other infectious agents that have been associated with conjunctivitis in rabbits include *S. aureus, Pseudomonas* species, *Haemophilus* species, *Treponema paraluiscuniculi*, mycoplasma, chlamydiae, and myxoma virus. In a colony of rabbits with chronic conjunctivitis, *Streptococcus pyogenes*, nonhemolytic *Escherichia coli*, *Coynebacterium pyogenes*, *Haemophilus* species, *Neisseria* species, and *Lactobacillus* species were isolated from conjunctival samples. Mucopurulent conjunctivitis and blepharitis with corneal ulceration have been associated with *S. aureus* infection in a rabbit.[28] Treatment with topical gentamicin ophthalmic ointment and systemic gentamicin was curative. Other causes of conjunctivitis in rabbits include foreign bodies, entropion, distichia, trichiasis, and high ammonia or dust content in the environment. Dental disease, including root elongation and dental abscesses, is also associated with conjunctivitis.

Unilateral or bilateral epiphora can be present in rabbits without conjunctivitis. The discharge often has a white, gritty appearance and may be intermittent and resistant to treatment with topical antibiotics. Root elongation of the maxillary incisors is a common underlying cause.[16] The elongated roots can cause an obstruction of the nasolacrimal duct at its flexure just caudal to the roots of the incisors. Radiographs of the skull are needed to assess the incisors; excess curvature of the incisor roots is abnormal. In one report describing two affected rabbits, radiographs revealed a cystic dilation of the nasolacrimal duct immediately caudal to the duct flexure, and the incisors were more arched than in normal rabbits.[24] Irrigation of the

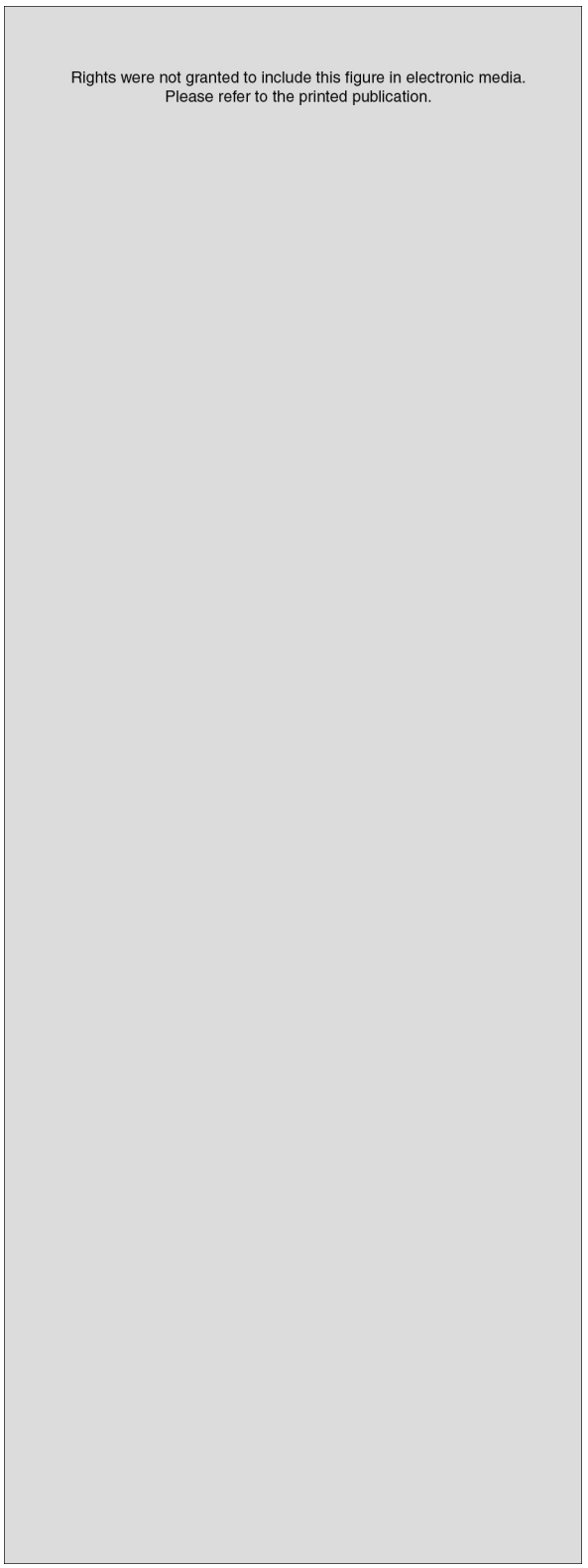

Figure 39-1 Diagram of the rabbit nasolacrimal duct. **A,** Lateral view with inset. The two sharp bends, the proximal maxillary bend (*pb*), and the bend at the incisor tooth (*ib*), are indicated. The inset shows the canaliculus (*C*) and the lacrimal sac (*S*). **B,** Dorsoventral view. *1*, Proximal portion of the duct extending from the punctum through the proximal maxillary curve; *2*, portion of the duct extending from the proximal maxillary curve to the base of the incisor tooth; *3*, portion of the duct extending from the base of the incisor tooth to the end of the lacrimal canal; *4*, distal portion of the duct extending from the end of the lacrimal canal to the nasal meatus. **C,** The nasal meatus of the nasolacrimal duct (*arrow*). The opening is enlarged for diagrammatic purposes. *(From Burling K, Murphy DJ, da Silva Curiel J, et al: Anatomy of the nasolacrimal duct and its clinical implications. Prog Vet Comp Ophthalmol 1991; 1:33-40.)*

nasolacrimal system in affected rabbits yielded opaque, white, gritty fluid. On cytologic examination, numerous macrophages, lipid-laden mesothelial cells, lipid droplets, and small numbers of bacteria and erythrocytes were present. The fluid cleared when ether was added, suggesting the presence of triglycerides or cholesterol. However, the role of lipid secretion in the pathogenesis of the obstruction was unknown. Bacteriologic culture of fluids used to irrigate the nasolacrimal ducts of both normal and affected rabbits yielded similar bacterial isolates; therefore, microorganisms apparently are not important in the pathogenesis of epiphora in rabbits. Bacterial isolates included *Staphylococcus, Pseudomonas*, *Moraxella,* and *Neisseria* species; *Oligella urethralis, Streptococcus viridans,* and *Bordetella bronchiseptica*. Inflammation was present in the nasolacrimal system of one rabbit.

In rabbits with epiphora, the diagnostic value of bacterial culture of the irrigation fluid is questionable. Skull radiographs are useful to detect underlying dental disease. Dacryocystorhinography using contrast material injected into the nasolacrimal system can help localize the site of obstruction, differentiate between a complete and partial obstruction, and identify any dilation.

Treatment of epiphora in rabbits can be frustrating. Irrigating the nasolacrimal duct is important to restore patency of the nasolacrimal system. After instilling a topical ophthalmic anesthetic, use a 23-gauge lacrimal cannula or a 24-gauge Teflon intravenous catheter to flush the duct (Fig. 39-2). Recurrence of the obstruction is common, and duct irrigation may need to be repeated every 2 to 3 days or weekly until a few consecutive clear irrigations are obtained. If topical antibiotic therapy is used, a broad-spectrum medication such as triple antibiotic solution is recommended. Topical nonsteroidal, anti-inflammatory ophthalmic medications, such as 0.03% flurbiprofen or 1% diclofenac, may help minimize irritation caused by the procedure. In rabbits with chronic or severe infections, concurrent topical ophthalmic and systemic antibiotic therapy may be needed. Suggested combinations include systemic enrofloxacin (Baytril, Bayer Corporation, Shawnee Mission, KS) and topical ciprofloxacin (Ciloxan, Alcon Laboratories, Inc., Fort Worth, TX) or gentamicin. In rabbits with evidence of underlying incisor root elongation, removal of the incisors can be considered in severe cases.

Cornea

Corneal dystrophy is the accumulation of cholesterol or lipid crystals in the cornea. This may develop spontaneously, as has been reported in American Dutch belted rabbits,[29] or from high dietary cholesterol. It also occurs in breeds that are predisposed to hypercholesterolemia, such as the Watanabe rabbit with heritable hyperlipidemia. In rabbits without systemic lipid

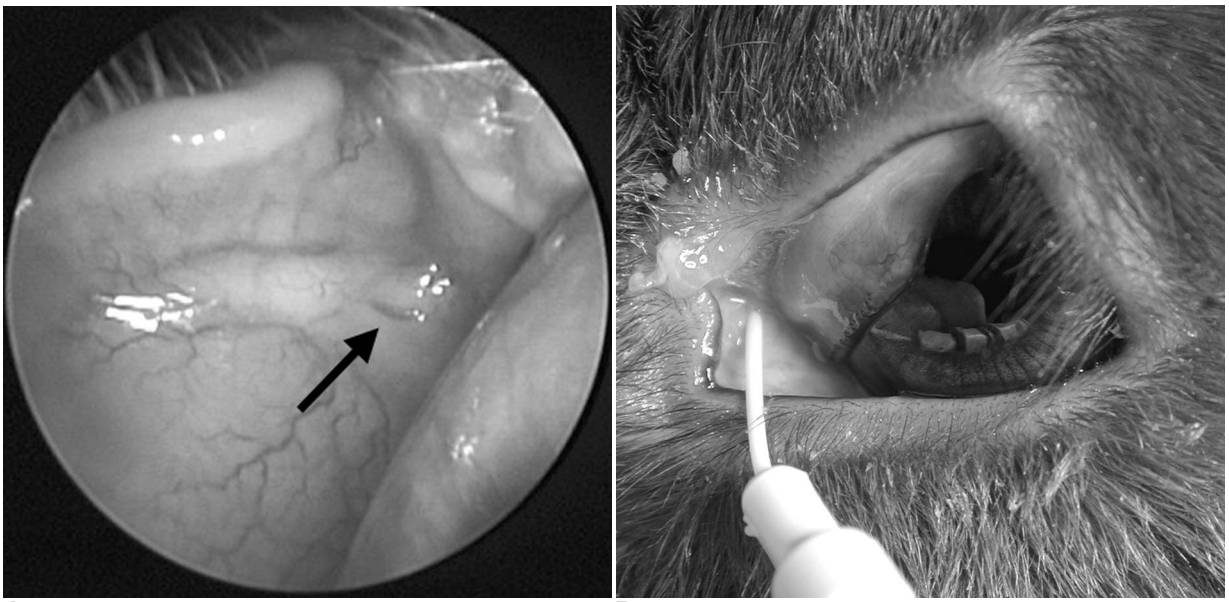

Figure 39-2 **A,** Rabbits have a single nasolacrimal punctum *(arrow)* in the ventral eyelid. **B,** Irrigation of the nasolacrimal duct in a rabbit with a 24-gauge Teflon intravenous catheter. *(**A,** Courtesy Susan Kelleher, DVM).*

abnormalities, spontaneous corneal dystrophy is usually bilateral and symmetrical and does not progress to visual impairment. In any rabbit with corneal dystrophy, carefully evaluate the fat content of the diet.

Progressive occlusion of the cornea with a conjunctival-like membrane is occasionally seen in rabbits.[12,35] Membranous corneal occlusion, or pseudopterygium, is a pain-free condition that may affect one or both eyes (Fig. 39-3). Ophthalmic examination reveals a circular membrane that originates at the limbus (the junction of the cornea and sclera) and gradually advances over the cornea. In severe cases, only a small central opening is present, allowing visibility of an otherwise normal globe. The membrane does not adhere to the cornea. The cause of this condition is unknown, although trauma has been suggested. Progressive membranous occlusion in rabbits has been compared with pterygium in humans. However, in humans the membrane is triangular and adherent to the cornea, whereas in rabbits it is nonadherent and circumferential from the limbus. Treatment with topical antibiotic or antibiotic-steroid medications has no effect. Surgically resecting the membrane and treating with topical antibiotics after surgery usually result in quick recurrence of the membrane. However, resecting the membrane a few millimeters beyond the limbus and treating with a topical antibiotic-steroid combination after surgery may prevent recurrence. Good results have also been obtained with surgical resection and the use of topical cyclosporine. Another described surgical technique is to incise the membrane into four quarters and suture each quadrant of the membrane to the inside of the eyelids.[35] With this technique, recurrence may be prevented for at least 1 year.

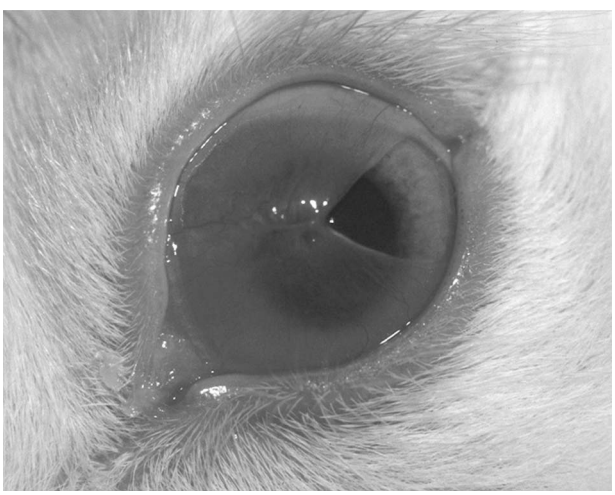

Figure 39-3 Progressive occlusion of the cornea with conjunctival-like tissue is present in this rabbit. The conjunctival-like tissue is not adhered to the cornea. The disease is not painful. *(Courtesy David Wilkie, DVM, MS.)*

Superficial, nonhealing corneal ulcers are occasionally seen in rabbits. Clinical signs are usually mild and include epiphora, conjunctival hyperemia, and blepharospasm. The ulcer is usually located in the paracentral cornea, is very superficial, and has redundant epithelial edges. The clinical appearance resembles an indolent ulcer, as seen in boxer dogs. Carefully examine the eyes of affected rabbits to eliminate potential causes such as abnormal hairs, lagophthalmos (inability to fully close the eyelid), facial nerve paralysis, or a foreign body. Treatment with a topical antibiotic solution or ointment usually fails to resolve the ulcer. Additional therapies such as corneal debridement, grid keratotomy, use of topical serum, application of corneal glue, tarsorrhaphy, or superficial keratectomy are usually necessary for the ulcer to heal.

Uveitis and Diseases of the Lens

Encephalitozoon cuniculi may cause granulomatous encephalitis and renal lesions in rabbits. Many rabbits infected with *E. cuniculi* are asymptomatic, but neurologic signs can include convulsions, tremors, torticollis, paresis, and coma. *Encephalitozoon cuniculi* infection has also been associated with phacoclastic uveitis. In one report, examination of phacofragmentation fluid from a rabbit revealed the presence of *E. cuniculi* DNA.[38] Most affected rabbits are young (less than 2 years), and dwarf rabbits appear predisposed to disease. Clinically, a white mass is often seen protruding into the anterior chamber (Fig. 39-4). Careful examination of the anterior segment of the eye with slit lamp biomicroscopy may reveal a break in the anterior lens capsule. The break is frequently hidden by inflammatory material and it may appear as if only the iris is involved in the inflammatory process. A focal cataract is often present in the area of the anterior lens capsule break. Signs of a severe pyogranulomatous anterior uveitis are usually present, such as conjunctival hyperemia, a swollen hyperemic iris, miosis, aqueous flare, and low intraocular pressure. The posterior segment of the eye is initially normal; however, if left untreated, severe uveitis and cataract formation can lead to blindness and possible phthisis bulbi or glaucoma. An abscess in the iris caused by *P. multocida* initially may resemble phacoclastic uveitis. Measuring serum antibody titers for *E. cuniculi* and *P. multocida* may aid in the differential diagnosis. Treatment of choice is surgical removal of the lens by phacofragmentation. Because of the rabbit's ability to regenerate a lens after this procedure, inserting an artificial lens after phacofragmentation is not recommended. Systemic treatment of *E. cuniculi* with albendazole (30 mg/kg PO q24h for 30 days, then 15 mg/kg PO q24h for an additional 30 days) has been reported.[38] More recently, fenbendazole (20 mg/kg q24h for 28 days) has proved effective in both preventing experimental *E. cuniculi* infection in rabbits and treating naturally infected, seropositive rabbits.[39] If the lens is not removed surgically, control of the uveitis with topical steroidal (such as 1% prednisolone acetate) and nonsteroidal anti-inflammatory medications as well as systemic fenbendazole or albendazole is necessary. Enucleation may be indicated if the uveitis cannot be controlled medically and a chronic painful eye is present.[46]

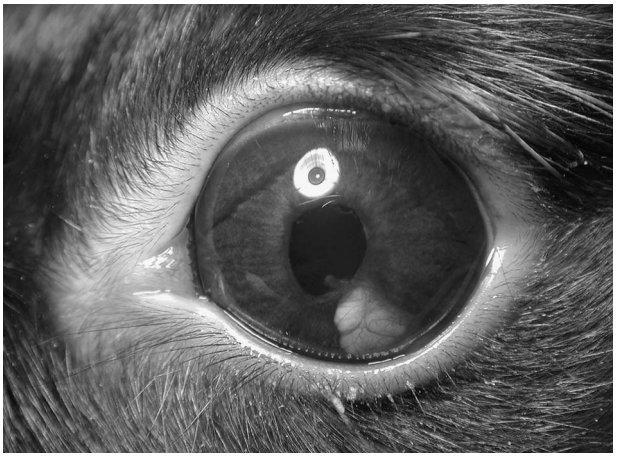

Figure 39-4 Rabbit infected with *Encephalitozoon cuniculi*. A white lesion is present in the iris, protruding into the anterior chamber. Lens involvement with cataract formation is present underneath the iridial lesion.

Glaucoma

Congenital glaucoma is inherited as an autosomal recessive trait in rabbits. In rabbits with glaucoma, the intraocular pressure is high as early as 3 months of age.[6] With increasing age, progressive buphthalmos with a markedly enlarged cornea, structural abnormalities of the iridocorneal angle, atrophy of the ciliary processes, and excavation of the optic nerve develop. Topical glaucoma medications used in dogs, such as 0.5% timolol maleate and 2% dorzolamide, may also be used in rabbits. Because response to therapy is unpredictable in rabbits, carefully monitor the intraocular pressure during treatment. Enucleation, inserting an intrascleral prosthesis, and laser cycloablation with a diode laser have also been used to manage glaucoma in pigmented pet rabbits. However, laser cycloablation cannot be used in albino rabbits. If left untreated in chronic cases, pressure-induced atrophy of the ciliary body may result in the intraocular pressure returning to normal.

Orbit

Retrobulbar disease processes are occasionally seen in rabbits. Clinical signs include progressive exophthalmos, protrusion of the third eyelid, and inability to retropulse the globe. Exposure keratitis may be present if the ability of the eyelids to close properly has been affected. Abscesses are a common cause of retrobulbar disease in rabbits, caused by infection with *P. multocida*[3] as well as other aerobic and anaerobic bacterial species. Dental disease with tooth root abscessation is often a predisposing factor, and a good dental examination and skull radiographs are indicated in any rabbit with a suspected retrobulbar mass (see Chapter 34). If available, a computed tomography scan is especially helpful in diagnosis (Fig. 39-5). Retrobulbar neoplasia is uncommon in rabbits.

An abscess in the retrobulbar space of a rabbit can be very difficult to treat. Because of the thick nature of the abscessed material, drainage of the abscess through the mouth, as performed in dogs and cats, may or may not be successful. If the abscess is caused by an abscessed tooth root, the tooth or teeth must be extracted to allow drainage and the rabbit must be treated with long-term systemic antibiotic therapy. In some cases, aggressive surgical debridement may be necessary. This may include exenteration of the orbit and sacrifice of a sighted eye. Even with aggressive surgical and medical management, the prognosis for recovery is always guarded. Anecdotal reports suggest some rabbits with retrobulbar abscesses respond to medical therapy with long-term (3 months) administration of benzathine/procaine penicillin G (for rabbits <2.5 kg, 75,000 U/rabbit SC q48h; for rabbits >2.5 kg, 150,000 U/rabbit SC q48h).[34]

Exophthalmos in obese rabbits may be caused by excessive fat deposition in the orbit. Periodic exophthalmos has been reported in rabbits with thymomas.[21,42] In one report, a localized myasthenia gravis associated with the thymoma was suggested as a cause.[42] More probably, the presence of a large intrathoracic mass obstructs blood flow in the right and left cranial vena cava, causing decreased vascular drainage from the head. Exophthalmos was also reported in a rabbit after chronic external jugular catheter placement[17] and has been seen clinically in a rabbit with

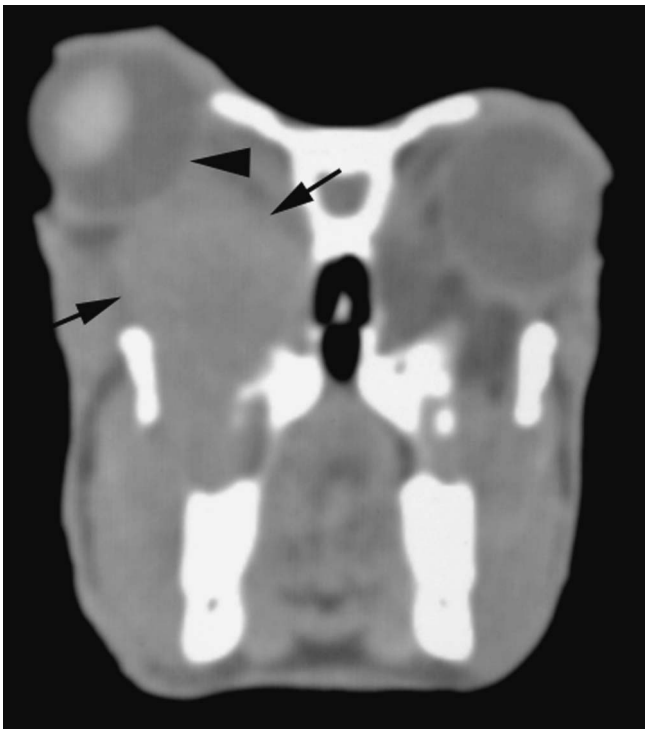

Figure 39-5 Computed tomographic scan of a rabbit with severe retrobulbar disease. A large mass (*arrows*) is present in the retrobulbar area, displacing the globe (*arrowhead*) dorsally and causing severe exophthalmos.

thrombosis of both external jugular veins after venipuncture and catheterization (K. Quesenberry, DVM, personal communication, 2002). Because the internal jugular veins of rabbits are relatively small, occlusion of the external jugular veins will compromise vascular drainage of the head. In the clinical case, the bilateral exophthalmos resolved spontaneously, presumably after the jugular veins became patent again (see Chapter 13).

Rabbits have several glands in the orbit. The lacrimal gland is located dorsolaterally, and the accessory lacrimal gland, divided into three lobes, is located along the caudal and ventral orbital margin. The superficial gland of the third eyelid is a small gland located near the cartilage of the third eyelid. The deep gland of the third eyelid, also known as the harderian gland, consists of two parts, a dorsal (white) and a ventral (pink) lobe. Prolapse of the deep gland of the third eyelid has been described in rabbits. Surgical correction with a pocket technique, as has been described in dogs, was successful in reducing the gland in one rabbit.[19]

FERRETS

Ferrets have prominent globes that are placed laterally in the skull, with very limited binocular vision. Their pupil is a horizontal slit and quickly responds to light. Topical 1% tropicamide may need to be applied to evaluate the fundus. Similar to dogs and cats, ferrets have a holangiotic retinal vascular pattern. The projection of retinal ganglion cells from the temporal area of the retina in albino ferrets differs from that of pigmented ferrets.[30] In pigmented ferrets, 6000 retinal ganglion cells project ipsilaterally to the brain, whereas in albino ferrets, only 1500 retinal ganglion cells project ipsilaterally. The significance of this difference has not been established.

Conjunctivitis in ferrets can be caused by viral or bacterial infection. Ocular signs of canine distemper virus, a fatal disease in ferrets, include mucopurulent oculonasal discharge, blepharitis, corneal ulcers, and keratoconjunctivitis sicca.[20] Conjunctival swelling and a proliferative lesion of the nictitans caused by infection with *Mycobacterium genavense* have been described in two ferrets. Other clinical signs in these ferrets included peripheral lymph node enlargement.[23]

Degeneration of corneal endothelial cells leading to progressive corneal edema and cloudiness of the cornea is seen in older mink (8-11 years).[15] Royal pastel females are predisposed. Unlike this disease in dogs, these mink do not develop corneal ulceration, pigmentation, or vascularization develop. There is no specific treatment for this condition, but symptomatic treatment with 5% sodium chloride solution or ointment 2 to 4 times a day may or may not improve corneal clarity.

A lymphoplasmacytic keratitis has been reported in a ferret with lymphoma.[33] An infiltrative lesion was present in the cornea that resembled corneal lesions reported in mink with Aleutian disease.

Cataracts are common in ferrets.[41] Progressive cataract formation has been reported in two genetically unrelated populations of ferrets.[26] In 1-year-old ferrets, cataracts were observed in 47% of animals examined. Severity ranged from clinically insignificant, small cataracts in the posterior cortex of the lens to blinding complete cataracts. By 18 months of age, cataracts were detected in virtually every animal, and in animals previously diagnosed, the cataracts had progressed. A genetically separate group had a combination of blinding cataract, microphthalmos, abnormal iris formation, and retinal detachment. In another ferret colony, microphthalmos, cataract, retinal dysplasia, and a persistent hyperplastic primary vitreous-type membrane were shown to be inherited as an autosomal dominant defect.[11] Dietary factors may play a role in the development of cataracts in ferrets. A diet high in fat or deficient in vitamin E or protein may promote cataract formation.[26]

Monitor the eyes of ferrets with cataracts regularly for the onset of secondary complications. Lens-induced uveitis can usually be controlled with topical 1% prednisolone acetate once or twice daily. Other complications caused by cataracts include lens subluxation or luxation and glaucoma. Ferrets that are blind because of cataracts usually adjust well in a home environment. However, cataract surgery can be performed successfully in ferrets. The lenses can be removed by phacofragmentation or by an extracapsular technique. Artificial lenses are not available in a size suitable for ferrets. Before cataract surgery, make sure the ferret becomes accustomed to frequent application of eye medications to facilitate easy treatment after surgery.

Retinal degeneration is seen in ferrets. Clinical signs are progressive loss of vision that may not be noticed until the disease is advanced. Ophthalmic examination reveals mydriasis with a very poor pupillary light reflex. Cataracts may or may not be present. Retinal vascular attenuation and tapetal hyperreflectivity are seen in the fundus. There is no treatment for retinal degeneration.

Lymphosarcoma is a common disease in ferrets. Although orbital involvement has only been reported in two ferrets,[25] it is occasionally seen clinically. Exophthalmos is often the presenting complaint. Ophthalmic examination reveals exophthalmos,

decreased retropulsion of the globe, and protrusion of the third eyelid. Lagophthalmos may result in exposure keratitis with corneal ulceration and vascularization. In the two ferrets described, lymphosarcoma was also detected elsewhere in the body.[25] Peripheral lymph nodes were affected in one ferret, and involvement of the liver, spleen, intestines, kidneys, and adrenals was present in the other.

The diagnosis of retrobulbar lymphosarcoma can be confirmed by cytologic examination of a sample from the retrobulbar area obtained by fine-needle aspiration. However, this procedure may be difficult because of the limited size of the retrobulbar space. Instead, suspicion of orbital lymphosarcoma may be confirmed by obtaining samples for diagnostic tests elsewhere in the body, such as a fine-needle aspirate or wedge biopsy of an enlarged lymph node. Therapy of retrobulbar lymphosarcoma is directed at treating the disease systemically with prednisone or chemotherapeutic agents. If the corneal epithelium is intact, protect the eye with lubricating ophthalmic ointment applied 2 to 4 times daily. If an ulcer is present, treat with an antibiotic ophthalmic ointment, such as triple antibiotic or gentamicin ointment, applied 3 to 4 times daily. While treating the ferret for lymphosarcoma, temporary tarsorrhaphy may be necessary to protect the cornea if pronounced exophthalmos is present.

Zygomatic salivary gland mucocele is another reported cause of exophthalmos in ferrets.[27] Fine-needle aspiration of a soft fluctuant swelling dorsotemporal to the eye yields a tenacious, blood-tinged fluid. Surgical excision is usually curative (see Chapters 3 and 12).

GUINEA PIGS

Guinea pigs have a paurangiotic retina that appears avascular on examination. Their eyelids are open from birth, and they have a rudimentary third eyelid.

Conjunctivitis is common in guinea pigs (Fig. 39-6). One common cause is *Chlamydophila psittaci*,[20] which causes a self-limiting disease manifested by mild chemosis, ocular discharge, and follicle formation. Cytologic examination of a specimen from a conjunctival scraping may reveal intracytoplasmic inclusion bodies in epithelial cells. Treatment is generally considered unnecessary. Vitamin C deficiency in guinea pigs causes conjunctivitis with a flaky discharge. Treatment is directed at correcting the dietary deficiency.

A spontaneous outbreak of listerial keratoconjunctivitis has been reported in hairless guinea pigs.[9] Clinical signs ranged from serous lacrimation with hyperemic conjunctiva to purulent, ulcerative keratoconjunctivitis with corneal neovascularization. *Listeria monocytogenes* was cultured from the ocular discharge. Treatment was not attempted.

Blepharitis caused by dermatophyte infection may be seen in young guinea pigs.[3] Topical antifungal therapy is usually effective.

Lymphosarcoma is rare in guinea pigs but has been reported to infiltrate the cornea.[37] Lymphosarcoma should also be considered as a differential diagnosis of conjunctival masses in guinea pigs.[2] Another differential diagnosis for conjunctival nodules in guinea pigs is a syndrome known as "pea eye." These nodules are protrusions of portions of the lacrimal or zygomatic glands and appear pale or pink. Treatment is not necessary because animals are usually not bothered by their presence.

A corneal dermoid has been reported in a guinea pig from a commercial colony.[31] A circular mass with a hair protruding from the surface was present on the central cornea. If noticed in a clinical patient, treatment by superficial keratectomy is recommended.

Cataracts have been seen in clinical patients. Cataracts can be removed surgically, but the procedure is difficult because of the small size of the globe, the large size of the lens, and the difficulty of intubating a guinea pig for general anesthesia.

Osseous metaplasia of the mesectodermal trabecular meshwork occurs in guinea pigs (Fig. 39-7).[14] Clinically, an arc of white, opaque material is visible in the anterior chamber, covering the iridocorneal angle. Vessels may be present overlying the osseous choristoma. Hematopoietic active bone marrow is present. This is usually an incidental finding and no specific treatment is necessary.

As in rabbits and other rodents, exophthalmos in guinea pigs may be related to dental disease. A tooth root abscess of a molar

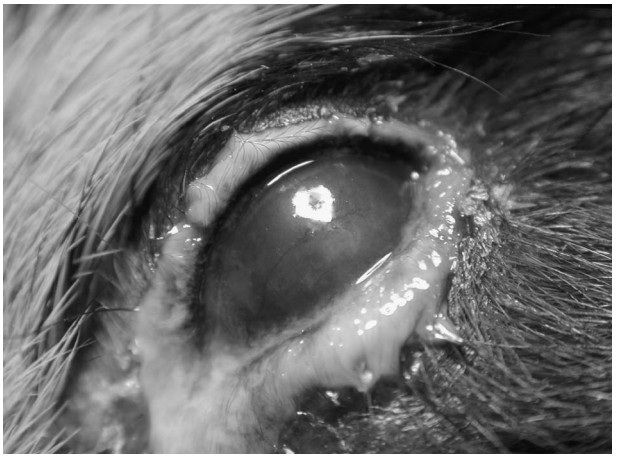

Figure 39-6 Conjunctivitis and keratitis in a guinea pig. Note the abundant mucopurulent discharge, corneal vascularization, and fibrosis.

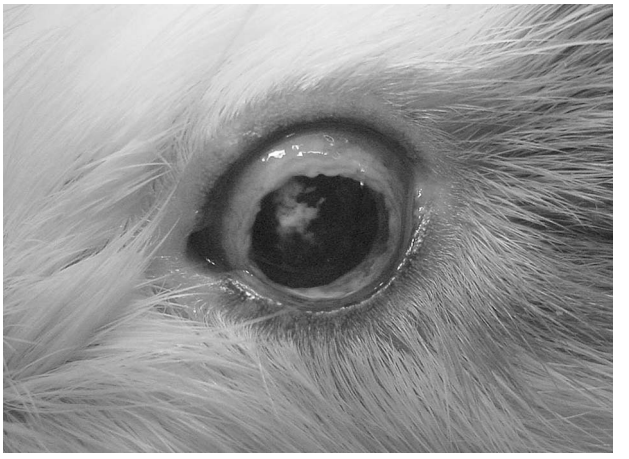

Figure 39-7 Osseous metaplasia of the mesectodermal trabecular meshwork in a guinea pig. White opaque material is present in the iridocorneal angle. *(From Brown C, Donnelly T: What's your diagnosis? Heterotopic bone in the eyes of a guinea pig. Lab Anim 2002; 31:23-25.)*

may result in maxillary sinusitis and orbital disease. Careful examination of the teeth is indicated in any guinea pig with exophthalmos.

CHINCHILLAS

Chinchillas have a vertical slit pupil and an anangiotic retina. Cataracts and asteroid hyalosis have been reported in older animals.[20]

The premolar, molar, and incisor teeth in chinchillas, as in guinea pigs, continue to grow and erupt throughout the animal's life. Insufficient wear can result in elongated roots of the premolar and molar teeth, resulting in progressive orbital disease including epiphora, decreased retropulsion of the globe, and proptosis (see Chapter 34). Computed tomography is more sensitive than radiographs in detecting early lesions.[10] The prognosis for advanced disease is poor.

RATS, MICE, AND HAMSTERS

The retina of rats, mice, and hamsters is holangiotic, with arteries and venules radiating from the optic nerve like spokes on a wheel. Rats have three lacrimal glands: intraorbital, extraorbital, and harderian.

Inbred strains of rats and mice are commonly used in commercial laboratories to study naturally occurring ophthalmologic diseases. Diseases involving all parts of the eye have been described. Common abnormalities include retinal degeneration, as in the RCS rat,[40] microphthalmos, and cataract. In addition to specific genetically determined ocular abnormalities in inbred strains, other spontaneous abnormalities occur. Ophthalmic examination of 6000 Sprague-Dawley rats revealed a focal linear retinopathy in 3% and a fundic coloboma in 0.5% of animals examined.[18] Spontaneous corneal degeneration has been described in Sprague-Dawley and Wistar rats,[4] and corneal dystrophy has been described in Fischer 344 rats.[22] Experimental infections also lead to ophthalmic abnormalities. Blepharitis with crust formation in the medial canthus and partial periocular alopecia were observed in mice experimentally infected with *Trypanosoma brucei*.[36]

Conjunctivitis in mice can be caused by numerous infectious agents, including *Pseudomonas aeruginosa, P. pneumotropica, Salmonella* species, *Streptobacillus moniliformis, Corynebacterium kutscheri,* Lancefield group C streptococci, *Mycoplasma pulmonis,* mousepox or ectromelia virus, Sendai virus, and lymphocytic choriomeningitis virus.[20] Bacteriologic culture and sensitivity testing may be indicated in individual rats and mice with persistent conjunctivitis. Epiphora in rats and mice can be caused by dental problems. Nasolacrimal duct obstruction can result from overgrowth or malocclusion of the incisors.

Chromodacryorrhea is red staining around the eyes seen in rats and mice. Inflammation of the harderian gland causes secretion of tears pigmented with porphyrin. Sialodacryoadenitis virus is a highly contagious coronavirus that replicates in the respiratory tract epithelium, causing rhinotracheitis, bronchitis, and alveolitis. The virus also causes sialoadenitis of the submandibular and parotid salivary glands and necrotizing dacryoadenitis of orbital and harderian lacrimal glands. Exophthalmos, epiphora, and keratoconjunctivitis may result. The infection usually resolves within 1 week in immunocompetent animals. In a study of athymic rats, infection persisted for more than 3 months, indicating that normal T-cell function is required for host defenses against the virus.[42] Infection with sialodacryoadenitis virus may also result in uveitis and multifocal retinal degeneration.[20] Complications from infection include corneal opacification, anterior and posterior synechiae, cataract, and glaucoma. Specific therapy is not available, and treatment is supportive only. Other causes for red tears include infection with parainfluenza virus type 3 or Sendai virus as well as pain or stress. Ammonia vapor from soiled bedding can act as an ocular irritant, predisposing animals to secondary infection. Keeping the housing areas well ventilated is important in preventing infection with sialodacryoadenitis virus.

Of clinical significance is the effect of xylazine on the lens in rats and mice. A reversible cataract has been observed after systemic use of xylazine. Transcorneal water loss and altered aqueous humor composition caused by corneal exposure have been suggested as a pathogenesis of cataract formation.[8]

In hamsters, keratoconjunctivitis can result from ammonia vapor from soiled bedding. Dental problems including tooth root infection may result in facial or retrobulbar abscesses, with hemifacial swelling, proptosis, and exposure keratitis as common sequelae. Treatment with systemic antibiotics is often unrewarding and such abscesses frequently result in death of the animal.[20]

Insidious globe enlargement with loss of vision has been reported in four hamsters.[13] Ophthalmic examination of these hamsters revealed an enlarged globe in both eyes, widely dilated pupils, lack of pupillary light reflex, a small optic nerve, and pale retinae. Histopathologic results suggested chronic open-angle glaucoma. Treatment of the suspected glaucoma was not attempted.

SUGAR GLIDERS

Sugar gliders have an avascular retina. Only a small residual tuft of fluorescein-impermeable vessels projects from the optic disc into the vitreous.[7]

Sugar gliders have prominent globes that are susceptible to trauma. Corneal ulcers may result from intraspecies fighting.[32] A retrobulbar abscess can result from a bite wound to the face or a molar root abscess. Corneal lipid infiltration may form in juvenile sugar gliders when the mother is fed a diet that is too high in fat. Although not reported, cataract formation is seen clinically.

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